THE PHILADELPHIA NEUROLOGICAL SOCIETY.

October 24, 1898.

The President, Dr. F. X. Dercum, in the chair.

A CASE OF TETANY.

Dr. Elizabeth R. Bundy presented a case of tetany from Dr. Spiller's clinic. The patient was a man twenty-two years of age, a native of Austria, who had been eighteen years in this country. He had been seized while on the street with violent spasms. These were frequent, beginning in the feet or hands, and attacking in turn nearly all the muscles of the extremities, as well as those of the head, chest and abdomen. The fingers were flexed upon the palm and the thumb upon the fingers, making a fist rather than the more usual "obstetrical hand." The forearms were flexed and the arms adducted, and toward the end of the spasm the claw-hand was produced for a moment or two. The toes were flexed, the feet were in the position of talipes equinovarus, and the entire lower extremity on each side was flexed and adducted. The face and forehead were also plainly involved in the spasms, which were so frequent and painful that an electrical examination was not made at this time. The electrical examination made later did not give abnormal reactions. The sensibility of the nerves was increased and slight pressure over the ulnar or external popliteal nerve caused numbness and tingling in the peripheral distributions. Trousseau's sign was easily obtained, but Chvostek's sign could not be elicited.

The diagnosis was based upon the character of the spasms, inasmuch as they were bilateral, intermittent, painful, beginning in the extremities and developing toward the trunk; on increased sensitiveness of the nerve trunks to pressure; on the presence of Trousseau's sign, which could be obtained at least two weeks after the spasms had ceased; on the presence of subjective sensory symptoms, namely, tingling of the skin and gastric irritability preceding the onset of the attacks for a day or two.

The patient gave a history of previous attacks during the past three or four years, occurring always in mild weather and lasting from three days to two weeks. No other member of the family seemed to be similarly affected.

Dr. William G. Spiller said he had seen quite a large number of cases of tetany in Vienna and had had the opportunity of observing carefully the case just described. He regarded it as undoubtedly one of tetany. Only two other conditions could be considered: hysteria and simulation. The occurrence of this disease in a person originally from Vienna, although he had been in this country eighteen years, the occurrence in a young male, and only in warm weather, were interesting statements. The spasms were somewhat atypical, but this type has been observed before. The involvement of the lower extremities and of the whole body is well known. The absence of increased irritability of the facial nerve and of the other motor nerves was uncommon, although the irritability of the sensory nerves was exaggerated.

Dr. Spiller thought that simulation need not be considered. The boy had never seen a case of tetany and knew nothing of the disease. Trousseau's sign is characteristic of tetany and was so marked in this case, that pressure over the large vessels and nerves of the arm caused a violent spasm of the muscles of the hand. The boy had stated that a fracture of a phalanx of one of the toes had been caused on one occasion by the attempt to straighten the toe while it was in spasm.

Dr. Joseph Sailer said he had seen a patient two years ago with tetany, who also had come from Vienna. The patient was a girl ten years of age and had been in this country two years. The symptoms were exceedingly mild in character. In the morning, she would have a slight cramp in the hand, soon disappearing and not recurring during the day. The characteristic signs were present, but slight. The diagnosis was confirmed by the electrical examination, which showed that the susceptibility of the nerves was greatly exalted. The patient passed from observation. She was one of a large family and none of the other members had ever shown symptoms of the disease.

Dr. James C. Wilson added another unpublished case. His patient was a young Frenchman thirty-two or thirty-three years of age, who after a series of business reverses went into ill health with gastro-intestinal catarrh and chronic diarrhea, and in the latter period of his illness developed well marked tetany. The symptoms of tetany continued for about two weeks, at the end of which time the patient died, chiefly in consequence of the intestinal trouble, but in part also from the exhaustion from the recurring attacks of spasm.

Dr. David Riesman said that about two years ago he had seen a case of tetany with Dr. Gordon Ross. The patient was a boy probably ten years old, a native of the United States. At first he appeared to be suffering from tetanus. There was, however, no reflex motor excitability, but most of the signs of tetany could be obtained. There was a marked hyper-extension of the foot with flexion of the great toe. The thyroid gland was believed to have been given without benefit. An attempt was made to find ptomains in the urine, as it was thought that the condition was due to gastro-intestinal intoxication, but on account of decomposition of the urine the search was discontinued.

With regard to tetany in dogs, Dr. Riesman said he had seen it a number of times. Dr. Carter and he in some experiments found that tetany usually developed within twenty-four hours after the removal of the thyroid gland. It has been held that this result could be prevented by not giving a diet rich in proteids. They attempted this in one case, but without result. It may be that the age of the dog has some influence. It was the experience of one of the pupils of Kocher that the age of the animal was a factor in the development of tetany.

A CASE OF ATAXIA LIMITED TO THE RIGHT ARM.

Dr. F. X. Dercum presented a woman of twenty-six years, who began to have numbness in the fingers and thumb of the right hand three months previously. Subsequently the fingers and hand became stiff, and she dropped various articles which she attempted to hold. A sense of stiffness and of dead weight subsequently and gradually spread up the forearm and arm as far as the shoulder. The right arm could be extended from the shoulder joint and moved freely in all directions, but the movements were awkward. The arm was readily flexed and extended at the elbow, but on attempting forcible extension of the biceps the arm was found to be less strong than its fellow. Movements of the wrist were performed freely in all directions; the extensors, however, were weaker than those of the opposite side. The grip of the right hand registered 22; that of the left hand 35. Movements of the fingers were executed freely, but were decidedly ataxic. The movements of the entire limb were ataxic. The tactile and temperature senses in the right limb were preserved, but the pain sense was slightly diminished over the forearm and fingers. The stereognostic sense in the right hand was lost. The patient complained of stiffness of the neck, and the movements of the neck were slightly restricted in all directions. More recently the left arm had begun to be a little weak. The sway was slightly exaggerated, but the gait was normal. The right knee-jerk was increased, and weak ankle clonus was present on the right side.

Note.—In a short time paralysis developed in all the extremities. The patient died about three weeks after she had been presented before the society. At the necropsy a tumor was found growing from the bone on the right side of the foramen magnum, and greatly compressing the medulla oblongata.

Dr. Charles K. Mills thought that this case was possibly cerebral and due to a focal lesion in the posteroparietal region of the cortex, or more probably to a lesion more deeply situated, but related in some way to the tracts which terminate in this region of the cortex or in the thalamus. It could hardly be a purely hysterical case, as many of the points developed were opposed to this view.

Dr. Charles W. Burr said that two years ago he had reported the case of a man who many years previously, after an accident to the head, had been palsied on the left side. He recovered from this palsy completely. After he was able to walk he chanced to put his hand into his pocket and found that he could not recognize what he took into his hand. He was told that this was a matter of no importance and that he would soon recover. That was fifteen years ago, and the condition still remains. When Dr. Burr saw the man there was no objective sign of disease except the scar. The patient was able to feel the lightest touch, but with his eyes shut he was unable to tell what position the arm or fingers were in, and could not recognize objects by touch.

Dr. Burr thought that the reason we are able to tell the shape of objects felt, is that we know the position that the hand must assume in order to grasp the object. In these cases the patients do not know what position the hands are in and hence do not recognize the shape of the object.

Dr. A. A. Eshner thought that the inability of Dr. Dercum's patient to determine the character of an object placed in her hand might be explained by a disturbance of peripheral sensory localization. If this girl could feel the object at each point where it touched her hand, she could probably determine its outline and arrive at a conclusion in regard to its form and size, but not necessarily as to its weight or its temperature.

Dr. F. X. Dercum said that if it were not for the marked rigidity of the neck and of the slight ataxia of the *left* arm, he would almost, without hesitation, have pronounced this to be a cerebral case. This rigidity might be due to a complicating rheumatic affection, or it might be due to a pachymeningitis. The iodides and mercurials had been used in this case and were well tolerated. This was very suggestive, of course, as pointing to possible multiple lesion.

A CASE OF BRAIN TUMOR.

Dr. J. L. Nicholson gave the history of a man who had had symptoms of brain tumor. In October, 1896, the patient fell and sustained a slight injury to his head. In the following December he fell down a stairway and injured the occipital region. He was slightly stunned for a few minutes. On February 14, 1897, while traveling in a railroad train, he felt a pain in his right leg and right arm, and became unconscious. On March 10, 1897, he had another attack of unconsciousness while on a train. On April 7 he had convulsions limited to the muscles of the right side of the body, lasting about three minutes, and he is said not to have lost consciousness at this time. The convulsions began in the right foot and leg, and then involved the trunk, arm, neck and face. The muscles of the right side, when the patient was examined, were found to be much weaker that those of the left. Ankle clonus and exaggerated reflexes were found in the limbs of the right side. No marked objective disturbance of sensation was noted, although the right leg and arm felt numb. No abnormal condition of the eye was found at this time, but one year later Dr. Risley noted optic atrophy of the outer side of each disk and signs of hemorrhages. The patient's memory was good until late in the disease, except shortly after one of his attacks. He had many right-sided convulsive attacks, and suffered from nausea and vomiting.

An autopsy was obtained August 28, 1898. A tumor was found slightly raised above the surrounding cortex, measuring 1½ inches anteroposteriorly and 1¼ inches vertically. It was situated at the upper portion of the fissure of Rolando on the left side of the brain, and dipped down into the longitudinal fissure ¾ of an inch. The tumor was not sharply defined from the surrounding cortex. Microscopically it was a round cell sarcoma.

Dr. Charles K. Mills said he had seen the man about a year and a half before the patient's death and had advised very strongly in favor of operation. He regretted that this advice had not been followed, as the case appeared to be an excellent one for operation.

Dr. William G. Spiller had seen this patient with Dr. Mills. Both Dr. Mills and he had urged an operation. The man had had many epileptic attacks, always limited to the right side, optic atrophy, weakness of the right side of the body, nausea, vomiting, headache and disturbance of the so-called muscular sense in the right upper and lower limbs. Dr. Spiller believed that the tumor was not very deep in the cortex. The epileptiform convulsions indicated a rather superficial lesion, and the paresis rather than paralysis, looked as though the growth had not penetrated very far into the motor cortex. The condition of the muscular sense, or sense of position, led him to believe that the parietal lobe was involved. He said that the supposed location of the muscular sense in the parietal lobe is of great interest, although we do not know that this view is correct. As the man could read it was not probable that the angular gyrus of the left side was invaded, provided we accept this gyrus as the center of word hearing.

Dr. Spiller reported a case which Dr. Lloyd and he had observed in the clinic of the former at the Philadelphia Hospital. A man, well advanced in years, complained during the night preceding his attack, of fatigue, headache and inability to sleep. In the morning he fell to the floor on attempting to rise from his bed, but was not unconscious. When he was found he had paresis of the left limbs and of the left side of the face and tongue. The left knee-jerk was exaggerated, and the left lower limb was rigid. Later the patient could raise his left upper limb above his head, but every movement of this limb was ataxic in an extreme degree, though the limb was not paralyzed. The mental condition of the patient prevented an examination of the condition of sensation.

At the necropsy, a hemorrhage was found in the right parietal lobe. The hemorrhagic area was about two centimeters in diameter and extended inward in the form of a cone to the lateral ventricle, having its base in the cortex. It was situated about four centimeters from the longitudinal fissure, and about two or three centimeters behind the Rolandic fissure. When Dr. Spiller saw the brain, it had been cut into frontal sections about a centimeter apart, and although the injury to the cerebral tissue prevented an exact determination of the location of the hemorrhage, it was evidently in, or very close to, the supramarginal gyrus.

Dr. Spiller thought that this case, together with the statements of v. Monakow and others, relative to the muscular sense, rendered the diagnosis of a cortical lesion in Dr. Dercum's case of ataxia in one arm not unreasonable.

Dr. Charles W. Burr and Dr. David Riesman reported a case of tumor of the brain, by which they attempted to show that acromegaly is due to disease of the pituitary body. (See p. 20.)

Dr. F. X. Dercum thought that the conclusions of Dr. Burr and Dr. Riesman would have been more completely established if they had reported a case in which the pituitary substance was all destroyed and there was no acromegaly. He could not see the close relation which this case bore to acromegaly, nor how it strengthened the view that disease of the pituitary body is the cause of acromegaly. It seemed to him further that the assumption that the pituitary body has different functions in different animals is a dangerous one. The pituitary body is a constant and fundamental structure in all vertebrates and presumably should possess equivalent values in all of them.

Dr. David Riesman said he might be able to answer one of the points raised by Dr. Dercum, namely, the failure to produce acromegaly in animals by the removal of the pituitary body. We cannot always apply the results of experiments on animal to man. For example, it is impossible to produce myxedema in dogs by the removal of the thyroid gland; we can produce tetany, but not myxedema, yet it will not be disputed that in man the absence or degeneration of the thyroid gland leads to myxedematous conditions.

What they attempted to show in their paper was merely this: that the cases of tumor of the pituitary body with no clinical signs of acromegaly cannot be directly used to controvert the view that disease of the pituitary body is the cause of acromegaly. If these cases had been examined with sufficient care to show that all of the pituitary body was destroyed and absent, then they might prove something. They might show that the total destruction of the pituitary body need not be followed by acromegaly and, as a corollary, that diesase of the pituitary body was not the causative lesion. Dr. Burr and Dr. Riesman had shown that in their case some of the pituitary body had remained and they had inferred that this was sufficient to carry on the functions of the gland. It might be said that the remaining part was too small to have any important function. We know, however, that the smallest piece of pancreas is sufficient to prevent glycosuria in animals, and that a very minute portion of the thyroid gland is sufficient to hold in abeyance the symptoms that follow the complete removal of that organ.

If we take the ground that acromegaly is due to the destruction of the pituitary body, some light is thrown on the function of the gland. The theory of its function that had suggested itself to Dr. Riesman might be formulated as follows: All tissues have a tendency to grow until a certain period when most of them cease in obedience, it has hitherto been said, to the law of growth. This law of growth is a vague term. The tissues grow by reason of some substance which normally stimulates them; they cease to grow, not because their power of growth is exhausted, but because of physical and chemical inhibitory influence. He believed that these inhibitory influences are chiefly chemical, and that in the case of the bones, for example, some substance accumulates in the body which checks the further growth of bone. This substance, he believed, is furnished by the hypophysis cerebri in minute quantities, probably from birth, and gradually accumulates until it is sufficient to counterbalance the inherent tendency of the bones, and perhaps of other tissues, to grow. When the pituitary body becomes diseased, and no more of this inhibitory substance is furnished, the bones, by virtue of their inherent tendency, become hyperplastic. Why this hyperplasia in acromegaly affects especially the peripheral parts of the skeleton and the face, he could not say, but this preference is not without analogy in other diseases.

The pituitary may have other functions beside those of inhibiting excessive growth of bony tissue.

Dr. Charles W. Burr believed that their case threw light on the subject of acromegaly. Cases of tumor of the pituitary body without acromegaly have been reported, and it has been believed from these gross examinations that disease of the pituitary body has nothing to do with acromegaly. This view is distinctly wrong. The sole object of their paper was to show that in order to prove that the pituitary body does not stand in a causal relation to acromegaly, it must be shown that in tumors of the pituitary body without acromegaly the substance of the organ has been completely destroyed by the new growth. When we recall that in fifty-seven out of sixty cases of acromegaly there was positive evidence of disease of the pituitary body, it must be admitted that there is some relation between the pituitary body and acromegaly.

Dr. Charles K. Mills reported a case of isolated paralysis of the ulnar nerve from unknown cause, and presented photographs.

CEREBRAL SYPHILIS AND SOME OF ITS MENTAL ASPECTS. A. E. Mink (Jour. of Am. Med. Assoc., 31, 1898, p. 5).

The author classifies the syndromes characteristic of brain syphilis as follows: 1. Syphilis of the base. 2. Syphilis of the vertex. 3. Syphilitic endarteritis which may be either basilar or vertical in situation. 4. Mixed forms, being both basilar and vertical in location and consisting usually of both specific endarteritis and meningitis. 5. Meningoencephalitis corticalis syphilitica. General symptoms common to nearly all varieties are: headache, especially in the earlier stages, and worse at night; insomnia and vertigo which may be complicated with nausea and vomiting. The patients are morose and irritable unless a syphilitic endarteritis predominates, in which case opposite conditions prevail, the stupor and drowsiness sometimes deepening into coma and even rendering the patient incapable of attending to the most pressing demands of nature. In many cases pronounced cardiac and gastro-intestinal irritability and a condition of vasomotor paresis of the cutaneous arterioles were observed.

The following phenomena are peculiar to both focal and diffuse forms of cerebral syphilis: 1. Sudden shifting of symptoms. 2. The predominance of cranial nerve palsies. 3. The multiplicity of palsies along with their coming and going. 4. The loss of particular accomplishments. 5. The concomitant appearance of symptoms due to destroying lesions of various cortical areas and the sudden coming or going of apparently fatal symptoms such as coma followed by perfectly lucid states. 7. The comparatively rapid termination in dementia. Mercury and the iodides are the sheet anchors in treatment, the former in the form of inunctions and the latter in some reliable syrup of hydriodic acid such as Gardner's, rather than the potassium or sodium salts, which, owing to the heavy dosage necessary, nearly always produce unpleasant symptoms, which may be entirely avoided by the use of the above preparation. VOGEL.